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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/734,281	12/11/2000	Marc Mercken	12546.4USC1	3720

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Attention of Mark T. Skoog
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EXAMINER
DUFFY, PATRICIA ANN

ART UNIT	PAPER NUMBER
1645	14

DATE MAILED: 06/26/2002

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.	Applicant(s)	
09/734,281	Mercken et al	
Examiner	Group Art Unit	
Duffy	1647	

—The MAILING DATE of this communication appears on the cover sheet beneath the correspondence address—

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE three MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, such period shall, by default, expire SIX (6) MONTHS from the mailing date of this communication .
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).

Status

☒ Responsive to communication(s) filed on 4-24-02.

☐ This action is **FINAL.**

☐ Since this application is in condition for allowance except for formal matters, **prosecution as to the merits is closed** in accordance with the practice under *Ex parte Quayle,* 1935 C.D. 1 1; 453 O.G. 213.

Disposition of Claims

☒ Claim(s) 20-29 is/are pending in the application.

Of the above claim(s) 25-28 is/are withdrawn from consideration.

☐ Claim(s) _____ is/are allowed.

☒ Claim(s) 20-24, 29 is/are rejected.

☐ Claim(s) _____ is/are objected to.

☒ Claim(s) 20-29 are subject to restriction or election requirement.

Application Papers

☐ See the attached Notice of Draftsperson's Patent Drawing Review, PTO-948.

☐ The proposed drawing correction, filed on _____ is ☐ approved ☐ disapproved.

☐ The drawing(s) filed on _____ is/are objected to by the Examiner.

☐ The specification is objected to by the Examiner.

☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119 (a)-(d)

☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 11 9(a)-(d).

☒ All ☐ Some* ☐ None of the CERTIFIED copies of the priority documents have been

☐ received.

☒ received in Application No. (Series Code/Serial Number) 08/106,758.

☐ received in this national stage application from the International Bureau (PCT Rule 1 7.2(a)).

*Certified copies not received: _____.

Attachment(s)

☒ Information Disclosure Statement(s), PTO-1449, Paper No(s). 4

☐ Interview Summary, PTO-413

☒ Notice of Reference(s) Cited, PTO-892

☐ Notice of Informal Patent Application, PTO-152

☐ Notice of Draftsperson's Patent Drawing Review, PTO-948

☐ Other _____

Office Action Summary

DETAILED ACTION

1. Applicants response filed 4-24-02 has been entered into the record.

Priority

2. The status of nonprovisional parent application(s) (whether patented or abandoned) should be updated. If a parent application has become a patent, the expression "now Patent No._____" should follow the filing date of the parent application. If a parent application has become abandoned, the expression "now abandoned" should follow the filing date of the parent application.

Information Disclosure Statement

3. The information disclosure statement filed 3-12-01 has been considered. An initialed copy is enclosed.

Election/Restriction

4. Claims 25-28 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected inventions, there being no allowable generic or linking claim. Election was made **without** traverse in Paper No. 13.

Double Patenting

5. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent

and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970);and, *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321© may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

6. Claims 20, 21, 23, 24 and 29 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-2 of U.S. Patent No. 6,238,.892. Although the conflicting claims are not identical, they are not patentably distinct from each other because the species as claimed is AT8 and the species anticipates the genus claims recited herein.

7. Claims 20, 22, 23, 24 and 29 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-8 of U.S. Patent No. 6,008,.024. Although the conflicting claims are not identical, they are not patentably distinct from each other because the monoclonal antibodies species as claimed is in the patents are AT180 and AT270 both bind phosphorylate epitope of tau and the species anticipate the genus claims herein.

8. Claims 20, 22, 23, 24 and 29 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-8 of U.S. Patent No. 5,843,779. Although the conflicting claims are not identical, they are not patentably distinct from each other because the monoclonal antibodies species as claimed is in the patents are AT120 binds a phosphorylate epitope of tau and the species anticipate the genus claims herein.

9. Given the number of different patent applications filed by the Inventors, Applicants should bring to the attention of the examiner any other pending applications or issued patents drawn to tau monoclonal antibodies.

Claim Rejections - 35 U.S.C. § 112

10. Claim 22 is rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a new matter rejection.

The recitation of claim 22 as "any variant peptide treated with a dephosphorylating agent" does not have conception by way of written description in the specification. Applicants amendment clearly broadens the scope of the narrow description set forth claim 3 as originally filed, therefore creating a new subgenus of variant polypeptides that were not contemplated in the specification as originally filed. As such, applicants amendment does not have conception, nor written description support in the specification as originally filed.

11. Claims 20-24 and 29 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for monoclonal antibodies which forms a complex with a

phosphorylated peptide YSSPGSPGT (SEQ ID NO:1) or YSSPGSPGT (SEQ ID NO:2) wherein said phosphorylated peptide is phosphorylated at the positions marked with * and specific species thereof, it does not reasonably provide enablement for a monoclonal antibody which ".... forms an immunological complex with any phosphorylated epitope present in a human abnormally phosphorylated tau protein .." variant peptides, or other phosphorylated epitopes. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The product claims recite that the monoclonal antibody ".... forms an immunological complex with a phosphorylated epitope present in a human abnormally phosphorylated tau protein .." and kit claims depend form the product claims. The specification exemplifies the monoclonal antibody AT8 which forms a complex with a phosphorylated peptide YSSPGSPGT (SEQ ID NO:1) or YSSPGSPGT (SEQ ID NO:2) wherein said phosphorylated peptide is phosphorylated at the positions marked with *. This monoclonal antibody has binds a phosphorylated epitope present in a human abnormally phosphorylated tau. However, the art teaches that the AT8 antibody does not in fact have the claimed properties of forming immunological complexes with variant polypeptides and not forming complexes with normal tau protein/phosphorylated epitopes treated with a dephosphorylating agent and variant peptides treated with a dephosphorylating agent and forming an immunocomplex with tau protein present in brain homogenates derived from human brain, the homogenates be isolated from a patient having died from a non-neurological disorder. Specifically, Goedert et al (Proc. Natl. Acad. Sci.. 90:5066-5070, 1993) that the AT8 monoclonal antibody binds human fetal and newborn rat brain tau, see Figure 1, page 5067. Clearly, since the AT8 antibody which forms a complex with other

human and rat proteins, the specification is not enabled for this specific claim language. The specification therefore fails to teach how to make antibodies which have the now recited property of forming "....an immunological complex with a phosphorylated epitope present in a human abnormally phosphorylated tau protein .. but selected to exclude...." as set forth in claim 22. Additionally, since AT8 forms a complex with a phosphorylated peptide YSSPGSPGT (SEQ ID NO:1) or YSSPGSPGT (SEQ ID NO:2) wherein said phosphorylated peptide is phosphorylated at the positions marked with * any other monoclonal antibodies which would also form a complex with a phosphorylated peptide YSSPGSPGT (SEQ ID NO:1) or YSSPGSPGT (SEQ ID NO:2) wherein said phosphorylated peptide is phosphorylated at the positions marked with *, would also do not have the claimed recited properties. Thus, the specification is not enabled for antibodies which have these recited functional properties and does not teach how to make such or describe a monoclonal antibody that binds an abnormally phosphorylated epitope that has such properties. The specification fails to teach other phosphorylated epitopes, which produce antibodies with the recited functional properties. Clearly, the AT8 antibody and similar antibodies which bind this sequence can not have the claimed properties of claim 22. Further, the specification teaches a sole epitope and does not point to other abnormally phosphorylated epitopes on tau. As such, applicants are not enabled for the breadth of the scope as is now claimed.

In the absence of further guidance from applicants as to other phosphorylated epitopes bound by monoclonal antibodies which have the claimed functional properties, one skilled in the art would be forced into undue experimentation to make and use monoclonal antibodies with these functional properties.

12. Claim 22 is rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

As to claim 22, the limitations in © and (d) are unclear because it is unclear what is excluded. For example, the monoclonal antibody is selected to exclude forming an immunological complex with a phosphorylated epitope treated with a desphosphorylating agent. It is unclear if the negative selection is against the phosphorylated epitope or the dephosphorylated epitope. A similar problem occurs with the limitation of (d). Applicants should make the negative exclusion clear, is the negative selection against the phosphorylated epitope or the resultant treated epitope that would not be phosphorylated ?

Claim Rejections - 35 U.S.C. § 102 or 103

13. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless --
> (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.
> (e) the invention was described in a patent granted on an application for patent by another filed in the United States before the invention thereof by the applicant for patent, or on an international application by another who has fulfilled the requirements of paragraphs (1), (2), and (4) of section 371© of this title before the invention thereof by the applicant for patent. The changes made to 35 U.S.C. 102(e) by the American Inventors Protection Act of 1999 (AIPA) do not apply to the examination of this application as the application being examined was not (1) filed on or after November 29, 2000, or (2) voluntarily published under 35 U.S.C. 122(b). Therefore, this application is examined under 35 U.S.C. 102(e) prior to the amendment by the AIPA (pre-AIPA 35 U.S.C. 102(e)).

14. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103© and potential 35 U.S.C. 102(f) or (g) prior art under 35 U.S.C. 103(a).

15. Claims 20, 23 and 24 are rejected under 35 U.S.C. 102(b) as being clearly by Dickson et al (Acta Neuropathol, 73:254-258, 1987 of record in parent prosecution).

Dickson et al teach a monoclonal antibody that recognizes a phosphorylated epitope in Alzheimer neurofibrillary tangles, neurofilaments, and tau proteins (see page 255, Results and Immunohistochemistry). As such, Dickson et al anticipates the claimed monoclonal antibody since the epitope to which the monoclonal antibody NP14 binds is phosphatase sensitive. Further, the epitope is present in tau protein and is present in Alzheimer disease (see page 254, column 2, first full paragraph). As such, the monoclonal antibody NP14 necessarily possesses all the binding characteristics claimed. The

differences in the methods of making the monoclonal antibodies as recited in the claims do not define over antibodies/hybridomas that have the same binding properties but are made by different methods. The production of a product by a particular process does not impart novelty or unobviousness to a product when the product is taught by the prior art. This is particularly true, when the properties of the product are not changed by the process in an unexpected manner. *In re Thorpe*, 227 USPQ 964 (CAFC 1985); *In re Marosi*, 218 USPQ 289, 292-293 (CAFC 1983); and *In re Brown*, 173 USPQ 685 (CCPA 1972). Therefore, even if a particular process used to prepare a product is novel and unobvious over the prior art, the product *per se*, even when limited to the particular process, is unpatentable over the same product taught by the prior art. *In re King*, 107 F.2d 618, 620, 43 USPQ 400, 402 (CCPA 1939); *In re Merz*, 97 F.2d 559, 601, 38 USPQ 143-45 (CCPA 1938); and *United States v. Ciba-Geigy Corp.*, 508 F.supp. 1157, 1171, 211 USPQ 529, 543 (DNJ 1979).

16. Claims 20, 23 and 24 are rejected under 35 U.S.C. 102(e) as being clearly by Trojanowski et al (U.S. Patent 5,601,985, issued Feb 11, 1997 with priority to August 14, 1991).

Trojanowski et al teach monoclonal antibodies that bind to a phosphorylated peptide which corresponds to residues 389-402 of human tau which was selectively phosphorylated at serine position 396, which they term T3P. Trojanowski et al disclose that anti-T3P antibodies were used immunocytochemically to stain tissue sections and in western blot experiments from Alzheimer's disease and control brains where the anti-T3P did not recognize normal tau (page 678, column 1, second paragraph). Trojanowski et al teach that dephosphorylating A68, to which the T3P antibody binds, provides for a drop in electrophoretic mobility with a treatment with a dephosphorylating agent and migrated to a position very close to that of dephosphorylated tau (page 678, column 2, Figure

2A).Trojanowski et al conclude that A68 is in fact derived from tau (see page 678, column 2, second full paragraph). Trojanowski et al teach test kits for diagnosing a disease comprising antigens capable of binding with antibodies reactive with a peptide comprising the sequence of LysSerProVal wherein the ser is phosphorylated or antibodies specifically reactive with the phosphorylated sequence (see column 8, first full paragraph). Trojanowski et al teach that the identification of abnormally phosphorylated tau can be accomplished by enzyme immunoassay (column 7, lines 40-45).

17. Claim 29 is rejected under 35 U.S.C. 102(e) as being clearly by Trojanowski et al (U.S. Patent 5,601,985, issued Feb 11, 1997 with priority to August 14, 1991) as applied to claims 20, 23 and 24 above and further in view of Dickson et al (Acta Neuropathol, 73:254-258, 1987) and Catty et al (Antibodies, Volume II A Practical Approach, IRL Press, at Oxford University Press, Oxford, 1990, pages 97-154).

Trojanowski et al is set forth supra. In particular, Trojanowski et al teach that the identification of abnormally phosphorylated tau can be accomplished by enzyme immunoassay (column 7, lines 40-45) and that kits for use in detecting such are contemplated. Trojanowski et al differ by not explicitly teaching a two site ELISA for detection of abnormally phosphorylated tau and its components in a kit.

Dickson et al teach a second monoclonal antibody binding a second phosphorylated epitope on tau.

Catty et al teach a variety of conventional formats for enzyme immunoassays (see page 103, Figure 2). In particular Catty et al teach the two site immunometric assay as a test for antigen where the capture antibody is attached to a solid phase such as a microtiter plate and the second antibody is labeled (see page 104-105). Catty et al teach

all the reagents and buffers needed to perform the ELISA (see page 101, Table 2, page 125 and pages 126-133).

It would have been *prima facie* obvious to measure abnormally phosphorylated tau in a sample by means of a two site indirect ELISA according to Catty et al by substituting the art established monoclonal antibodies of Trojanowski et al and Dickson et al in the method as a means of detection of abnormally phosphorylated tau because Trojanowski et al teach that the identification of abnormally phosphorylated tau can be accomplished by enzyme immunoassay already in the art and commercially available (column 7, lines 40-45). Further, it would have been prima facie obvious to assemble the all of the necessary reagents (antibody attached to the microtiter plate, buffers, substrates, and developing agents) in a microtiter kit format for convenience and economy for the consumer and to reduce overall processing time for the assay by providing a reduced number of steps (i.e. binding the antibody to the microtiter plate well).

18. Claims 20, 22, 23 and 24 are rejected under 35 U.S.C. 103(a) as being unpatentable over Lee et al (Science, 251:675-678, February 3, 1991) in view of Goding (Monoclonal Antibodies, Academic Press Inc., London 1983, pages 56-97).

Lee et al discloses raising antisera in rabbits to a phosphorylated peptide which corresponds to residues 389-402 of human tau which was selectively phosphorylated at serine position 396, which they term T3P. Lee et al disclose that anti-T3P antibodies were used immunocytochemically to stain tissue sections and in western blot experiments from Alzheimer's disease and control brains where the anti-T3P did not recognize normal tau (page 678, column 1, second paragraph). Lee et al teach that dephosphorylating A68, to which the T3P antibody binds, provides for a drop in electrophoretic mobility with a treatment with a dephosphorylating agent and migrated to a position very close to that of

dephosphorylated tau (page 678, column 2, Figure 2A). Lee et al conclude that A68 is in fact derived from tau (see page 678, column 2, second full paragraph). Lee et al is silent with respect to the development of monoclonal antibodies to this phosphorylated epitope. The differences in the methods of making the monoclonal antibodies as recited in the claims do not define over antibodies/hybridomas that have the same binding properties but are made by different methods.

Goding teaches routine methods of making monoclonal antibodies with defined immunogens.

It would have been prima facie obvious to one having ordinary skill in the art at the time that the invention was made to use the teachings of Lee et al to generate a specific monoclonal antibodies using only the conventional techniques of Goding et al because of the well established advantages of high-affinity, high specificity and unlimited supply that are central to monoclonal antibodies. One would have been motivated to make monoclonal antibodies to decrease the lot to lot variability that can happen with polyclonal antisera. One of ordinary skill in the art would have a reasonable expectation of success give the demonstrated immunogenicity of this phosphorylated tau epitope.

19. Claims 20, 21, 23 and 24 are rejected under 35 U.S.C. 103(a) as being unpatentable over Dickson et al (Acta Neuropathol, 73:254-258, 1987 of record in parent prosecution) in view of Kosik et al (Neuron, 1:817-825, 1988) and Binder et al (J. Cell. Biol., 101:1371-1378, October 1985).

Dickson et al teaches using extracts form Alzheimer brain as the immunogen for raising monoclonal antibodies which recognize a phosphorylated epitope present in tau.

Dickson et al are silent as to the method used for raising their monoclonal antibodies and the epitopes on tau to which their antibodies bind.

Kosik et al disclose that tau protein has been shown to be an integral component of Alzheimer's paired helical filaments (PHF), they disclose the immunogens used for raising monoclonal antibodies to tau and they disclose a method of defining the antigenic sites that monoclonal antibodies bind within human tau (Figure 3 on page 820). They mapped epitopes for 5 monoclonal antibodies that span almost the entire length of tau suggesting that PHF contain tau in its entirety or nearly in its entirety (Abstract on page 817 and page 822, column 1, second full paragraph) and includes the epitope of monoclonal antibody tau 1 which binds to Pro Lys Ser Gly Asp Arg Ser Gly **Tyr Ser Ser Pro Gly Ser Pro Gly Thr** Pro Gly (Figure 3) that includes the dephosphorylated epitope of claim 22 in bold. Kosik et al disclose that the tau 1 epitope is phosphatase sensitive which has led to the proposal that the molecule might undergo potentially significant conformational changes as the result of the addition or removal of phosphate. Tau 1 recognize NFT only after treatment with alkaline phosphatase as such has been considered to recognize a site that is aberrantly phosphorylated during the process of NFT formation (page 822, column 2, first full paragraph).

Binder et al disclose producing monoclonal antibodies that binds to tau polypeptides by immunizing mice with bovine microtubule-associated protein, MAP-2, fusing splenocyte with SP/O myeloma cells and testing the resulting hybrid cells form those which produce monoclonal antibodies which bind MAP-2 or tau. One antibody Tau-1 was cross-reactive with rat brain, exhibiting binding to several polypeptides in the tau region of the gel. Binder et al disclose subcloning this cell line then injecting it into a mouse for the production of antibody-containing ascites fluid where cells obtained from the ascites fluid

were harvested, cultured and subcloned. Binder et al disclose using monoclonal antibodies in ELISA formats including a competitive format for quantitative determination of tau (page 1372, column 2, third full paragraph).

It would have been prima facie obvious to one having ordinary skill in the art to make additional monoclonal antibodies which bind to phosphorylated epitopes on tau as alternates to the ones taught by Dickson et al for detecting phosphorylated tau and NFT in Alzheimer's disease using the conventional methods as taught y Binder et al because Binder et al teach conventional methods of making hybridoma cell lines which produce monoclonal antibodies and isolated monoclonal antibodies from mouse ascites fluid which bind to tau protein using a variety of immunogens including bovine MAPs, bovine tau, detergent extracts of rat brain protein and Alzheimer basal forebrain and methods for detecting tau using a competitive ELISA format and Dickson teaches that monoclonal antibodies raised against extracts from Alzheimer brain samples recognize a phosphorylated epitope on tau and are used to immunostain brain samples from patients with Alzheimer's disease. Thus, one would have reasonably expected to make other hybridomas with produce monoclonal antibodies which bind to phosphorylated epitopes on abnormally phosphorylated tau present in Alzheimer brain tissue as the functional equivalents of the monoclonal antibody AT8 using known immunogens and conventional methods. One would have been motivated to screen for those monoclonal antibodies which recognize the phosphorylated epitope to which the Tau-1 antibody when that epitope is abnormally phosphorylated as an aide to detecting and diagnosis brain as a means of simplifying Kosik assays because Kosik teaches epitopes on tau which are recognized by monoclonal antibodies including the Tau-1 epitope which is phosphatase -sensitive and only reveled after treatment with alkaline phosphatase, and thus by using an antibody which

recognizes the phosphorylated epitope one could eliminate the need to treat the sample with alkaline phosphatase prior to antibody binding. It would have been obvious to one of ordinary skill in the art to assemble the reagents in a kit format because kits are convenient and economical means of providing the necessary reagents for the user. One would have been motivated to immobilize those monoclonal antibodies on a solid support for isolating abnormally phosphorylated tau protein from brain samples taken from patients with Alzheimer's disease because affinity purification is conventional used in the art as a means for isolating antigen from a sample.

Status of Claims

20. No claims are allowed.

21. Any inquiry of a general nature or relating to the status of this general application should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Papers relating to this application may be submitted to Technology Center 1600, Group 1640 by facsimile transmission. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). Should applicant wish to FAX a response, the current FAX number for Group 1600 is (703) 308-4242.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Patricia A. Duffy, Ph.D. whose telephone number is (703) 305-7555. The examiner can normally be reached on Monday-Thursday and Saturday from 10:30 AM to 7:00 PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Lynette Smith, can be reached at (703) 308-3909.

Patricia A. Duffy, Ph.D.
June 25, 2002

Patricia A. Duffy, Ph.D.
Primary Examiner
Group 1600